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M L. Potter

Lisa M. Tokach

Steven C. Henry

See next page for additional authors

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Authors

M L. Potter, Lisa M. Tokach, Steven C. Henry, Joel M. DeRouchey, Michael D. Tokach, Raymond R. Rowland R, Richard D. Oberst, Richard A. Hesse, Robert D. Goodband, Jim L. Nelssen, and Steven S. Dritz

GENETIC BACKGROUND INFLUENCES PIG GROWTH RATE RESPONSES TO PORCINE CIRCOVIRUS TYPE 2 (PCV2) VACCINES[1]

M. L. Potter[2], L. M. Tokach[3], S. S. Dritz[2], S. C. Henry[3], J. M. DeRouchey, M. D. Tokach, R. D. Goodband, J. L. Nelssen, R.R.R. Rowland[2], R. D. Oberst[2], and R. A. Hesse[2]

Summary

A total of 454 pigs (21 d of age, 13.4 lb) were used in a 130-d field study to investigate porcine circovirus type 2 (PCV2) vaccine effects on growth performance of boars and gilts of 4 different genetic backgrounds: A×A (Duroc-based sire and dam), B×B (synthetic line sire and dam lines derived from Duroc, Pietrain, and Large White), A×B, and B×A.

Pigs were identified as potential test pigs at birth and ear tagged for identification. Characteristics including litter, genetic background, gender, and birth weight were recorded and used in allotting PCV2 vaccine treatment groups. Pigs were vaccinated according to label dose with a 2-dose commercial PCV2 vaccine (Circumvent PCV, Intervet Inc., Millsboro, DE) at weaning (d 0) and again 14 d later. Vaccinated and control pigs were comingled within the same pen for the duration of the study. Pigs were individually weighed on d 0, 40, and 130 to measure growth rate. Backfat and loin depth were measured on d 130 by using real-time ultrasound. Blood was collected on d 0, 40, and 130 for indirect fluorescent antibody measurement of PCV2 antibodies and polymerase chain reaction (PCR) analysis for determination of PCV2 virus load.

By d 130, vaccinates were heavier ($P < 0.01$) than controls. However, the magnitude of the weight difference between control and vaccinates was almost 4 times greater in the A×A pigs than in the B×B pigs ($P < 0.05$). On the basis of growth performance, the different genetic backgrounds responded differently to the PCV2 vaccination even though they were comingled in the same pen. In the 2 pure-line populations, even the best performing portion of the population appeared to benefit from vaccination, suggesting that growth performance of most pigs is being affected by PCV2 infection.

Control pigs exhibited a late increase in PCV2 antibody levels, a consequence of natural infection. In contrast, vaccinated pigs did not exhibit a late-finisher antibody rise. Vaccinated pigs possessed a decreased viral load (as quantified by PCR PCV2 viral DNA) at both d 40 and 130. The data demonstrate that genetic background affects either the expression of porcine circoviral disease or the response to the PCV2 vaccine.

Key words: circovirus, genetics, growth, PCV2, swine, vaccination

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[2] Department of Diagnostic Medicine and Pathobiology, Kansas State University.
[3] Abilene Animal Hospital, PA, Abilene, KS.

Introduction

The primary agent of porcine circoviral disease (PCVD) is porcine circovirus type 2 (PCV2). The approved case definition for PCVD defines a PCVD herd as one which demonstrates one or more of the following clinical manifestations: wasting, respiratory or enteric signs, high mortality, porcine dermatitis and nephropathy syndrome, or reproductive disorders. Porcine circoviral disease is confirmed by finding microscopic lesions consistent with the disease in affected pigs as well as the presence of viral antigen in tissues.

Reported risk factors associated with PCVD include litter of origin, management factors, and gender as well as genetics. Only limited, controlled research has been completed to define the role of these risk factors in the development and expression of PCVD and response to vaccination. The focus of this study was to further elucidate the contribution of genetic background to PCVD by comparing the response of different genetic lines of pigs to PCV2 vaccination in a high-health herd with naturally circulating PCV2.

Procedures

A 1,700-sow multiplier farm in Kansas was used for this field study. This single-site farm maintains a high-health status; it is porcine reproductive and respiratory syndrome virus negative and without evidence of *Mycoplasma hyopneumoniae* infection since its stocking in 2000. Despite the high-health status, the presence of PCV2b virus had been documented in this herd. However, the primary concern was an increase in morbidity characterized by ill-thrift and slow growing pigs as mortality was within the expected historic range on this farm.

For this 130-d study, a total of 454 pigs from 4 genetic backgrounds were ranked by birth weight within litter and gender (boar or gilt) and randomly assigned to PCV2 vaccine treatment group (vaccinated or nonvaccinated control). Birth weight was balanced across vaccine treatment. Genetic background included pure lines of A×A (Duroc-based sire and dam) and B×B (synthetic line for the sire and dam primarily derived from Duroc, Pietrain, and Large White) and crossbreds A×B and B×A.

Vaccine treatment pigs were vaccinated intramuscularly with a 2-dose commercial PCV2 vaccine (Circumvent PCV, Intervet Inc, Millsboro, DE) according to label dose at 21 and 35 d of age. Vaccinated pigs were comingled with nonvaccinated control pigs for the duration of the trial.

Pigs were individually weighed and bled at birth, weaning (d 0), end of nursery (d 40), and off test (d 130). Loin depth and backfat measurements were collected by using real-time ultrasound on d 130.

Removals and deaths were recorded during all phases. There were 6 deaths in the nursery phase, 25 deaths in the finisher phase, and 6 other records removed because of data entry errors or unrelated physical defects. Data analysis was performed on the 417 pigs that had complete growth records.

Comparisons between vaccinates and nonvaccinated controls, genetic background, and gender were made in a 2 × 4 × 2 factorial treatment design. Litter of origin was managed as a random effect. Statistical analysis was performed by using the Proc GLIMMIX procedure of SAS to obtain least square means and standard errors for the response criteria.

Results

Despite active PCV2 infection, there was no discernable pattern of mortality among the genetic backgrounds, and overall mortality was similar between vaccinates (6.8%) and controls (7.0%). There were no 3-way gender × genetic background × vaccine interactions found for the response criteria in this study with the exception of backfat depth after

weight adjustment. The significant interaction ($P = 0.02$) was the result of control A×B crossbred boars having a higher weight-adjusted backfat depth than vaccinated A×B crossbred boars (11.9 ± 0.4 mm for controls vs. 10.9 ± 0.4 mm for vaccinates). Within all other gender by genetic background combinations, backfat depth was similar between controls and vaccinates.

Weaning age and weight and off-test age were not different for the vaccine × genetic background least squares means (Table 1). A vaccine × genetic background interaction was noted for nursery ADG, finisher ADG, and overall wean-to-finish ADG ($P = 0.05$, $P = 0.05$, and $P = 0.04$, respectively). In the nursery phase, this interaction was due to the A×B vaccinates having lower ($P = 0.04$) ADG than A×B controls. In contrast, B×A and B×B vaccinates had numerically higher ADG than controls. For the A×A pigs, controls and vaccinates had similar ADG. Therefore, in the nursery period of this study, genetic background did affect how vaccinates performed compared with controls; controls demonstrated higher nursery ADG in a single genetic background, whereas in the other lines, there was little to no performance difference between vaccinates and controls. Although the interaction was statistically significant, we are unsure of the biologic significance. In the finisher phase of production, ADG was lower ($P < 0.01$) for the A×A controls than for the control pigs of A×B, B×A, and B×B. In contrast, A×A vaccinates had similar ADG to all groups except A×B ($P = 0.04$). Thus, the magnitude of the difference between control and vaccinate groups was greater for the A×A pigs than for B×B and crossbred pigs. Wean-to-finish ADG followed a pattern similar to that of finisher ADG.

At d 130, A×A control pigs weighed less ($P < 0.01$) than controls from the other 3 genetic backgrounds, whereas A×A vaccinates weighed less ($P = 0.04$) than B×A vaccinates.

Prior to adjusting backfat and loin depths to a common off-test weight, it appeared there were significant differences in loin depth and numerical differences in backfat measurement between vaccine treatment groups. After adjustment, there was a genetic background effect ($P < 0.01$) for loin depth as well as a 3-way gender × genetic background × vaccine interaction ($P = 0.02$) for backfat measurement. Despite the 3-way interaction for backfat depth, there was no significant effect of vaccination on backfat ($P = 0.62$) or loin depth ($P = 0.29$) after adjustment to a common off-test weight.

Indirect fluorescent antibody analysis demonstrated antibody responses to both vaccine and natural PCV2 exposure (Figure 1). In response to vaccination, vaccinates had increased ($P < 0.01$) antibody production by d 40 compared with controls, but as a result of natural PCV2 exposure, controls demonstrated a rise ($P < 0.01$) in antibody levels compared with vaccinates by d 130. PCV2 DNA template copies per reaction provided an estimate of viral load (Figure 2). PCV2 vaccination reduced mean viral load in vaccinates compared with controls at both d 40 ($P < 0.01$) and 130 ($P < 0.01$)

Discussion

The results from this study demonstrate that genetic background affects response to PCV2 vaccination or PCVD expression as measured by growth rate. The findings in this study are unique because this herd did not fit the accepted case definition for PCVD; yet, this study clearly demonstrates that PCV2 vaccination improved the growth performance of vaccinated pigs compared with controls.

The difference in magnitude of the finisher ADG and wean-to-finish ADG was 3 and 5 times greater, respectively, in A×A pigs than in B×B pigs. In addition, within each crossbred genetic background, vaccinated pigs consistently had numerically increased ADG compared with controls. Vaccinated pigs were

19.7, 6.2, 10.2, and 5.0 lb heavier compared with nonvaccinated controls for A×A, A×B, B×A, and B×B genetic backgrounds, respectively. Similar to other studies we have conducted, PCV2-vaccinated pigs demonstrated increased growth rate during the finisher phase. However, the magnitude of the weight difference was almost 4 times greater in the A×A pigs than in the B×B pigs. Although the B×B pigs grew faster than the A×A pigs, they had a similar overall pattern of weight distribution about their means. There is a right shift in the off-test weights of vaccinates compared with controls in each population (Figures 3 and 4). This indicates that within each of these genetic backgrounds, all the vaccinated pigs had increased growth rate. Even in apparently clinically unaffected pigs, the PCV2 virus appears to affect growth rate.

Carcass composition was not affected by vaccination in this study after adjusting for off-test weight. Genetic background, however, did affect carcass traits. Pigs from the Duroc-based lines had decreased loin depth and increased backfat compared with pigs from the Duroc-, Pietrain-, and Large White-based lines.

There was a clear rise in antibody production by d 40 due to vaccination as indicated by the higher PCV2 antibody titers in the vaccinates compared with controls. A rise in antibody titer in the control pigs from d 40 to 130 indicated active PCV2 infection due to field virus exposure during the trial period. In contrast, vaccinates had a decrease in PCV2 antibody titer from d 40 to 130, which suggests that vaccinates have increased resistance to infection. The results of this study support previous research findings that PCV2 vaccination effectively decreases viral load, even under comingled conditions. Controls had a larger quantity of viral templates per reaction compared with vaccinates at d 40. By d 130, the difference between the treatment groups remained; however, mean template copies per reaction were reduced to 3.8 for controls compared with 1.3 for vaccinates. The biologic significance of these viral load quantities remains to be determined; however, the potential for the PCV2 vaccine to aid in the reduction of viremia and viral shedding is apparent.

The data in this study demonstrate that genetic background affects either the expression of PCVD or the response to the PCV2 vaccine, as measured by growth performance. Therefore, genetic background should be considered a risk factor for expression of PCVD or a factor that affects response to PCV2 vaccine.

Table 1. Effect of PCV2 vaccine treatment and genetic background on ages, weights, growth rates, and carcass characteristics[1]

	Genetic background[2]									
	A × A		A × B		B × A		B × B			Vaccine × Genetic
Item	Control[3]	Vacc.	Control	Vacc.	Control	Vacc.	Control	Vacc.	SE[4]	Probability, $P <$
no. of pigs	62	55	60	65	34	32	55	54		
Age, d										
Weaning	21.2	21.1	20.3	20.3	21.3	21.3	19.7	19.6	0.6	0.71
Off test	151.5	151.4	150.6	150.6	151.7	151.7	150.0	150.0	0.7	0.41
Weaning weight, lb	12.8	13.5	13.8	13.9	14.5	14.2	12.8	13.2	0.7	0.51
ADG[4], lb										
Nursery phase	0.84^{a}	0.85^{abc}	0.96^{bd}	0.90^{ace}	0.96^{bcde}	1.02^{de}	0.92^{abcde}	0.88^{abc}	0.05	0.05
Finisher phase	1.70^{a}	1.91^{b}	1.93^{b}	2.02^{c}	1.91^{bc}	2.00^{bc}	1.88^{b}	1.95^{bc}	0.05	0.05
Wean-to-finish	1.44^{a}	1.59^{b}	1.63^{bc}	1.68^{bc}	1.63^{b}	1.71^{c}	1.60^{bc}	1.60^{bc}	0.05	0.04
Off-test weight, lb	200.9^{a}	220.6^{b}	226.7^{bc}	232.9^{bc}	226.7^{bc}	236.9^{c}	220.7^{b}	225.7^{bc}	6.5	0.05
Carcass characteristics, mm										
Backfat depth	11.4	12.0	12.1	12.0	11.2	11.7	10.6	10.8	0.5	0.46
Loin depth	59.2	62.2	65.7	66.9	66.3	69.0	68.8	69.6	1.3	0.32
Backfat depth (weight adjusted)	12.2	12.1	11.9	11.6	11.1	11.2	10.7	10.7	0.5	0.79
Loin depth (weight adjusted)	62.3	62.6	65.1	65.4	65.8	67.1	69.2	69.2	0.9	0.82

Note. Results reported as least squares means.

^{abcde} Within a row, means without a common superscript letter differ ($P < 0.05$).

[1] A total of 454 pigs from 4 genetic backgrounds were assigned to vaccine treatment by ranking them by weight within litter and gender and randomly assigning each pig to either vaccine or nonvaccinate control, balanced by birth weight across vaccine treatment. Pigs were individually weighed at birth, weaning (d 0), end of nursery (d 40), and off test (d 130). Backfat and loin depth were measured at d 130.

[2] Genetic backgrounds used were A×A (Duroc-based sire and dam), A×B, B×A, and B×B (synthetic line for the sire and dam primarily derived from Duroc, Pietrain, and Large White).

[3] Vaccine treatments included vaccinates (2 cc Circumvent PCV, Intervet Inc., Millsboro, Delaware) and nonvaccinated controls. Vaccine was administered intramuscularly at 21 and 35 d of age.

[4] SE among treatment groups differed because of unbalanced design. In this table, the highest SE among the treatment groups was reported.

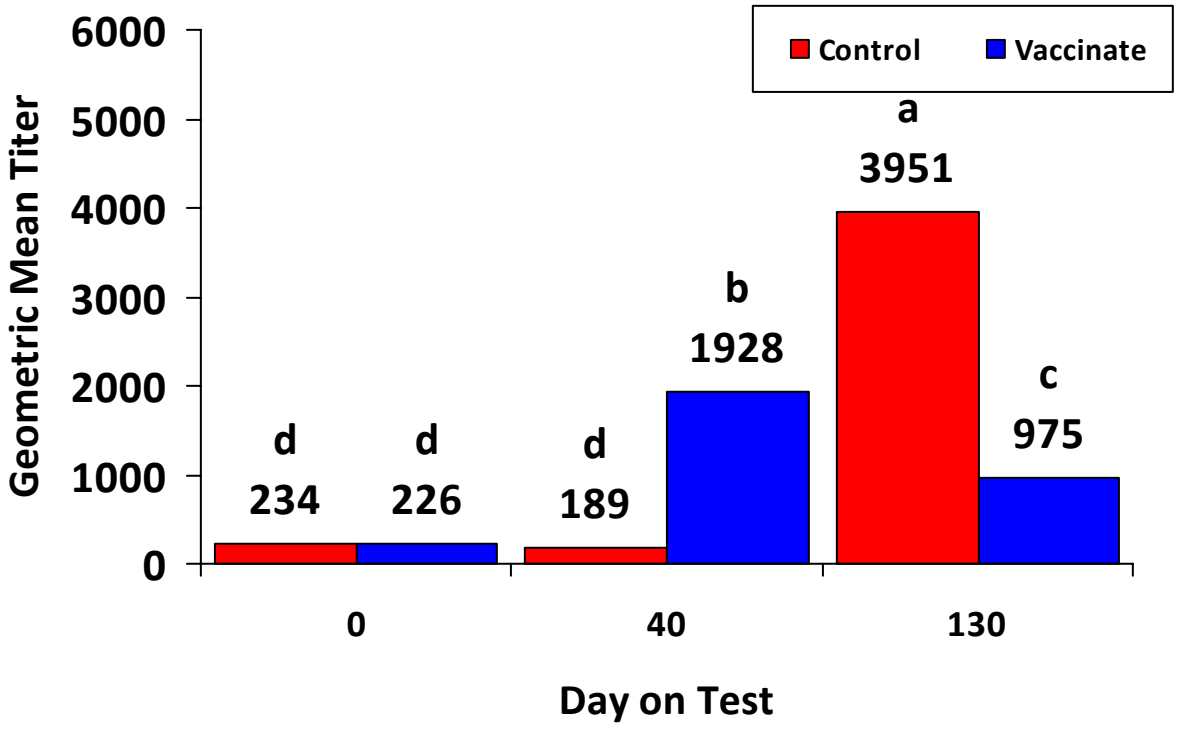

Figure 1. Effect of PCV2 vaccination and time on indirect fluorescent antibody geometric mean titer (vaccine × time $P < 0.01$; a,b,c,d $P < 0.01$).

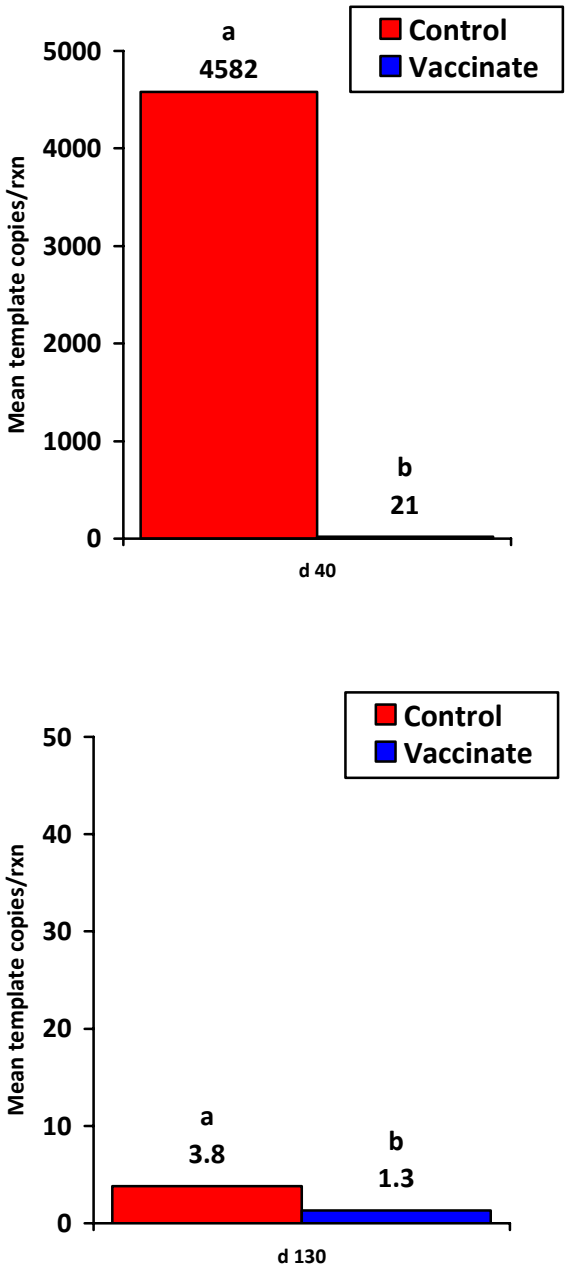

Figure 2. Effect of PCV2 vaccination and time on PCV2 viral template quantity (a,b $P < 0.01$ within day).

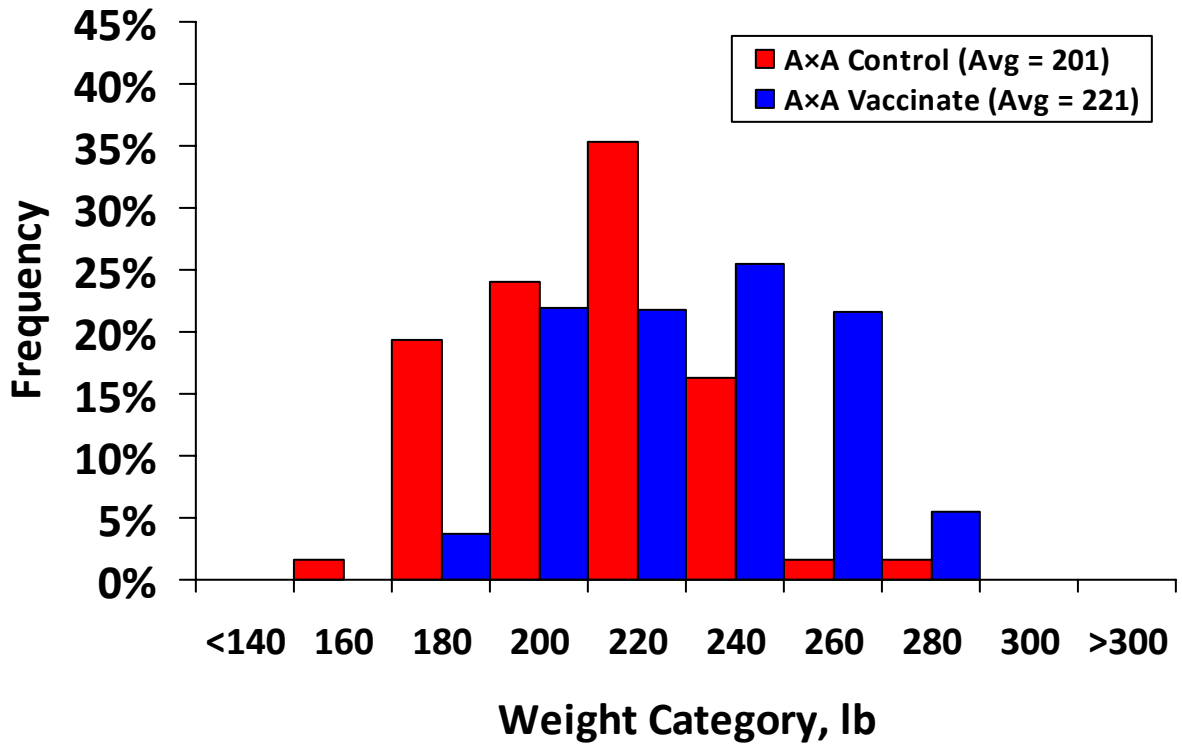

Figure 3. Distribution of off-test pig weights for control vs. vaccinated pigs of genetic background A×A.

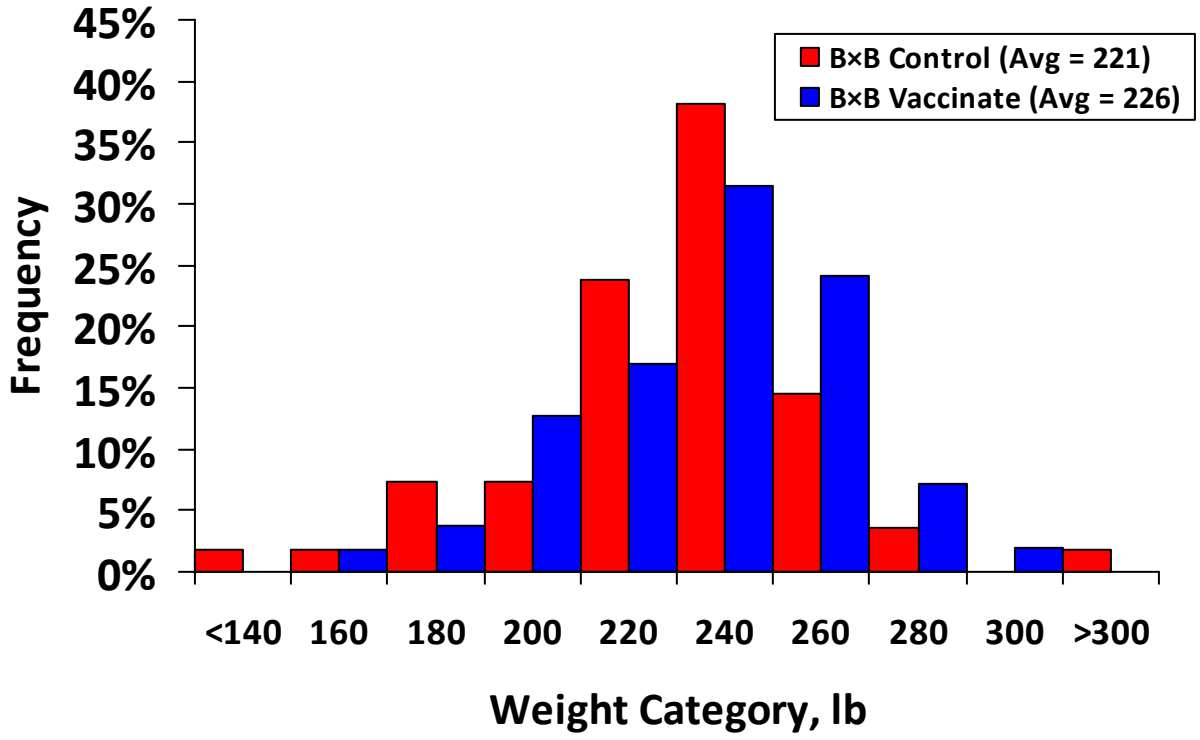

Figure 4. Distribution of off-test pig weights for control vs. vaccinated pigs of genetic background B×B.